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December 21, 2006

Frederick R. McGowen, Esq.
Goodwin Procter, LLP
599 Lexington Avenue
New York, New York 10022

Re: Harbir Singh v. Herbalife International Communication, Inc. et. al.

Dear Mr. McGowen:

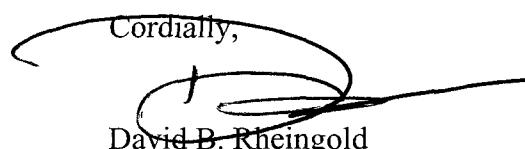
Enclosed is Dr. Lawrence W. Shields' December 4, 2006 report. Attached is his curriculum vitae and his testimony list. He does not have any published materials.

Dr. Shields' fees are as follows:

1. \$500.00 for a report;
2. \$500.00 per hour for EBT this includes preparation and travel time; and
3. \$7,500.00 (minimum) - court appearances.

We expect to receive an updated testimony list and we will forward it to you.

Thank your for your attention on this matter.

Cordially,

David B. Rheingold

DBR:lb
Enclosures

LAWRENCE W. SHIELDS, M.D.
Board Certified Neurologist

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December 4, 2006

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New York, New York 10016-3042

Re: Harbir Singh
DOE: 05/10/03

Gentlemen:

At your request, I examined Harbir Singh in my office on November 14, 2005, in the presence of his wife, Doina Caragata. I have also reviewed the following available medical records concerning Harbir Singh who suffered a subarachnoid hemorrhage on May 10, 2003.

St. Vincent's Medical Center - 05/10/03 admission record;
St. Vincent's Medical Center - 06/27/03 inpatient rehabilitation record;
St. Vincent's Medical Center - Outpatient Clinic records 2003-2005;
Manhattan Neurosurgical Associates, P.C. by Alan Hirschfeld, M.D. -

Office note dated 07/30/03

Fourth Amended Generic Expert Report of Steven R. Levine, M.D.

United States District Court

Southern District of New York

In Re: Ephedra Products Liability Litigation

Deposition of Doina Caragata

United States District Court

Southern District of New York

In Re: Ephedra Products Liability Litigation

My qualifications, case summary, citation of relevant clinical data, case analysis and conclusions follow.

QUALIFICATIONS

I received my undergraduate degree from Princeton University in 1961. I attended New York University College of Medicine and was awarded a Doctor of Medicine in 1965. I performed my internship at Kings County Hospital, Brooklyn, New York from July 1965 through July 1966. I was a Resident in Neurology at Mount Sinai Hospital, New York from July 1968 through June

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1971. Between internship and residency, I served as a General Medical Officer and then Chief Medical Officer at the United States Public Health Service Hospitals in Lompoc, California and Danbury, Connecticut. I am licensed to practice medicine in New York, Pennsylvania and California. I became Board Certified in Neurology by the American Board of Psychiatry and Neurology in 1976. I have served as the Acting Chief of Neurology at Brookdale Medical Center, a seven hundred plus bed general acute care hospital, from 1971 to 1986. There, I had the sole ultimate responsibility for maintaining the standard of neurologic practice in all areas, including stroke and stroke management in addition to my teaching and administrative responsibilities.

I have been and continue to be involved in the clinical practice of Neurology since 1971 and have also served as Clinical Director of a large medical (but predominantly neurologic) group. My responsibilities as Clinical Director in this setting also included monitoring and maintaining the standard of medical care as practiced by the group. Throughout my medical career I have been consistently heavily involved with the diagnosis and treatment of patients with strokes and have maintained the same standards in private practice.

The following opinion is formulated based upon my qualifications, as enumerated above, review of the medical literature, including medical journals, text books and published case reports and my personal experience in treating the full spectrum of neurological disorders. Specifically, in the case of Harbir Singh, I base my opinions on personal experience in the evaluation and treatment of strokes and stroke related disorders and neurovascular disorders, including imaging studies of cerebrovascular disease. This experience and my knowledge gleaned from the literature constitutes an informing and relevant basis for my expert opinion.

CASE SUMMARY

On May 10, 2003, Mr. Singh, then a 41 year old right handed man was admitted to St. Vincent's Catholic Medical having suffered a severe headache with nausea that morning. Additionally, it is reported that he had passed out, fell and hit his head. Emergency head CT scan at St. Vincent's Catholic Medical Center revealed subarachnoid hemorrhage. A neurosurgical consultation performed at 2:15 p.m. described Mr. Singh as being stuporous with mild left sided weakness and a recorded blood pressure of 175/118. Mr. Singh had taken Herbalife*, an ephedra containing compound on May 10, 2003, the day of his subarachnoid hemorrhage and had been ingesting this product daily for the previous year. Mr. Singh used three green pills twice a day per the instructions of the distributor, to promote weight loss.

*The product label reads: each tablet contains 21 mg of concentrated ephedra extract and 3 mg of caffeine.

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Mr. Singh denied any significant previous illness and had not received medical care prior to this incident. Mr. Singh had been a pack a day smoker for 30 years, usually imbibed a cup of tea and 2 alcoholic drinks per day. He states he did not smoke on the day of his subarachnoid hemorrhage.

As mentioned above, a head CT scan performed on 05/10/03 demonstrated subarachnoid hemorrhage. On admission, Mr. Singh was rated as having a Hunt-Hess Grade of III (clinical grade) and Fisher Grade IV (distribution of hemorrhage) subarachnoid hemorrhage. Mr. Singh was intubated, placed on a subarachnoid hemorrhage regimen including Nimodipine and coils were placed in the intracranial left internal carotid artery aneurysm he harbored. On May 12, 2003, an arteriogram and endovascular intracranial electro-thrombosis of a left internal carotid artery bifurcation aneurysm were performed. The aneurysm was described as 7 x 5.4 mm with a neck diameter of 4 mm. The aneurysm was bilobar with a posterior projecting fundal teat. The arteriogram described probable fibromuscular dysplasia (FMD) of the left internal carotid artery. No evidence of vasospasm was recorded in the body of the angiogram report, however, under "impression", evidence of vasospasm was noted.

Laboratory studies on May 10, 2003:

Abnormal results: WBC 12.2 (h); MCH 31.9 (h); Mean platelet Vol 7.3 (l); Neutrophil 73.3 (h); lymphocyte 19.8 (l); monocyte 3.9 (l); Abs Neu 8.90 (h).

General Chemistry: Sodium serum 130 (l); protein 4.7 (l); albumin 2.6 (l); AST 59 (h); urea 21 (h); calcium 7.9 (l); ALT 98 (h).

Urinanalysis: UA protein 100 AB.

Normal results: Hgb 15.8; Hct 44; RRC 4.9; PT 12.7; INR .96; APPT 25.2; Glucose 80.

Tox Screen: Amphetamine Ø; Barbiturates Ø; Benzodiazepine Ø; cocaine Ø; opiates Ø.

Echo report of 05/21/03. Normal LV functions. No clot/veg seen; LAE; LVH; trace MR; no AI..

During his hospitalization, Mr. Singh underwent a ventriculostomy, tracheostomy and PEG tube placement. His hospital course was complicated by the development of non-communicating hydrocephalus, aneurysmal re-bleeding, intraparenchymal bleeding, ischemic injury to the right

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lenticular nucleus, meningitis secondary to ventriculostomy infection and hyponatremia. Mr. Singh was transferred to St. Vincent's Inpatient Rehabilitation facility on June 27, 2003, where he received multi-disciplinary rehabilitation including physical therapy for gait dysfunction. Mr. Singh was discharged on July 9, 2003, with improved gait and balance requiring close supervision climbing stairs. Medication on discharge included Phenobarbital 60 mg to prevent seizures. On July 20, 2003, Mr. Singh was seen by Dr. Hirschfeld for post-operative neurosurgical follow-up and was found to have "no noticeable unsteadiness of gait, no focal neurologic deficits and was able to return to work". Mr. Singh, however, states he was never able to return to work, but occasionally accompanies his wife to the jewelry store where she works.

REVIEW OF SELECTED MEDICAL RECORDS

ST. VINCENT'S HOSPITAL
ADMISSION: 05/10/03 DISCHARGE: 06/27/03

05/10/03 2:15 p.m. - Neurosurgery Attending Admitting Note

" ... 41 y/o man ... sudden onset ... H/A today associated w/ nausea, vomiting ... altered mental status .. Mild left sided weakness ... arousable ... followed commands ... drift back to sleep when not stimulated ... intubated ... PMH: unknown ... doesn't go to doctor ... Meds: none ... allergies, penicillin ...CT: ... diffuse local cistern SAH greatest in (L) sylvian fissure ... possibly ... illeg ... defect in ...illeg... fissure .. Mild to moderate hydrocephalus ... no intracerebral clot ... some blood in ventricular system ...Exam: ... currently being intubated ... 3 mm and reactive pupils ... BP: 175/118 on admission now 159/ arousable to tactile stimulation, verbalizes, knows name ... followed simple commands ... moves all 4 ext w/ focal motor deficit ... responded to pain ... DTRs equal ... IMP: H&H Grade III SAH possibly from ruptured (L) PCA aneurysm, mild hydrocephalus possibly w/ elevated ICP ... Plan: transfer from ER to NICU, intubate, sedate, keep on ventilator, Dilantin, Nimodoprin , Diprivan drip ... ventriculostomy today ASAP - possibility of angio and coiling today..." Signed, A. Hirschfeld.

05/10/03 Neurosurgery Attending Procedure Note

" ... 4:15 p.m. ... ventriculostomy ... hydrocephalus ... altered mental status... required ventriculostomy as emergency life saving procedure ... ventriculostomy placed ... OP moderately high ... CSF bloody tinged ... going for angiography and possibly coiling of aneurysm...". Signed, A. Hirschfeld.

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05/10/03 Report of CT Scan of Head - Incomplete report

“... 8:34 p.m. ... IMP: intrnal placement of coil in suprasellar cistern to the (L) midline, internal placement of a shunt catheter ... mild reduction in the overall size of the ventricular system ...”. Unsigned note ... page 2 of report not present in the medical record

05/12/03 Report of Bilateral Internal Carotid Bilateral Vertebral Intracranial Cerebral Arteriography

“... Findings: ... 7.0 x 5.4 mm posteriorly projecting bilobed left internal carotid bifurcation oaneurysm with a 3.7 mm neck. A fundal teat is seen at the dome of the aneurysm ... medial lenticulostriate vessels ... extremely close to medial side of aneurysm arising from A1 ... lateral lenticulostriate ... illeg ... slightly more lateral to aneurysm on M1 segment ... no other intracranial aneurysm identified ... There is a peculiar appearance of the cervical (L) internal carotid artery ... appearnace of dysplasia ... likely fibromuscular dysplasia ... does not have ... classic string of pearl appearance ... no fenestrations or other intracranial abnormalities ... no evidence of vasospasm ... IMP: dysplastic cervical left internal carotid artery which is fibromuscular dysplasia.; left internal carotid bifurcation aneurysm bilobed projecting posteriorly with a fundal teat directed posteriorly and superiorly; maximum sagittal diameter of aneurysm is 7 mm; transverse width of aneurysm at its maximum is 5.4 mm ... 3.7 mm neck is right at internal carotid original slightly eccectric towards the A1 side ... evidence of vasospasm is seen (sic). No other intracranial aneurysm identified at this time...”. Signed, Bruce Zablow, M.D.

05/12/03 Report of Neuroendovascular Surgery

“... Procedure: endovascular intracranial electrothrombosis of (L) internal carotid cerebral bifurcation aneurysm intraoperative and postoperative (L) internal carotid cerebral arteriography ... Procedure & Findings: ... there was greater than 95% packing of the aneurysm w/ excellent packing of the dome ... no ... downstream embolization of the anterior middle cerebral vessels ... no evidence of dissection ... in the internal artery in the neck ... no evidence of vasospasm ... presence of a 7 mm x 5.4 mm (L) internal carotid bifurcation aneurysm w/ multiple lobes ... fundal teat projecting superiorly and posteriorly ... aneurysm has a 3.7 ... neck ... Post Procedure Dx: (L) internal carotid intracranial bifurcation aneurysm ... 7.0 mm x 5.4 w/ neck diameter of 4 mm ... dysplasia of the cervical (L) internal carotid artery ... probability of fibromuscular dysplasia ... no other intracranial aneurysm identified ... no evidence of vasospasm see at the time ...”.

05/13/03 CT Scan of Head w/o Contrast

“ ... the ventriculostomy catheter remains w/in the (R) lateral ventricle ... the ventricles are decompressed ... small about of blood in the atria of both ventricles w/ small amount of blood in the 3rd ventricle ... blood from the 4th ventricle has cleared. There is still visible blood in the basal cisterns and the (L) sylvian fissure and some cortical sulci over the (L) hemisphere... small

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amount of hemorrhage along the course of the ventriculostomy catheter in the (R) frontal lobe. There is also some hemorrhage in the (R) basal ganglia and (R) thalamus. The surrounding edema about the small area of hemorrhage ... There are GDC coils in the (L) internal carotid bifurcation area, there is now new subarachnoid hemorrhage or hemorrhage in the proximity of the coils to suggest aneurysm re-rupture...”.

05/17/03 CT Scan of the head w/o Contrast

“ ... there has been an interval decrease in the density of the (R) frontal hemorrhage w/ increase in surrounding edema. There is a small amount of air w/I the hemorrhage and a new hemorrhage w/I the (L) high frontal lobe extending from the centrum semiovale to the corpus callosum ...”.

05/24/03 CT Scan of the Head

“... lucencies throughout the lentiform nucleus on the (R) side ... likely ischemic in etiology ...”.

06/03/03 CT Scan of the Head

“... compared to prior study dated 05/24/03, there has been increase in the size of the ventricles resolution of the acute hemorrhage w/I the (L) frontal lobe ... the remainder of the study is unchanged ...”.

General Discharge Summary

“ ... D/C Meds: Phenobarbitol 60 mg p.o.? T.i.d....”.

06/27/03 Attending Admitting Note

“... 41 y/o man admitted to St. Vincent's on 05/10/03 secondary to severe HA ... had to be intubated ... head CT showed SAH ... cerebral angiogram showed a (L) internal carotid artery aneurysm ... pt underwent endovascular coiling on 05/10/03 ... also S/P ventriculostomy, S/P tracheostomy, S/P PEG placement ... meningitis (acinetobacter/corynebacterium factor) secondary to ventriculostomy infection... Echo WNL ... Exam: ... alert, awake ... in no acute distress ...”. No signature

07/01/03 Rehabilitation-Psychology Note

“ ... 4:30 p.m. ... presented w/ subdued constricted affect but was receptive to support. Pt spoke slowly but had no overt communication difficulty ... Pt acknowledged feeling shocked and shaken up by recent events as he had never been in the hospital before. At the same time, he experienced hope in the future ... born and raised in Indian... BA degree ... worked in business ... married 3-1/2 years ago ... has been working w/ a friend in an import/export fabric business in the Village... Strong religious faith ... feels his prayers were answered in terms of his recovery so far ... expressed thinking ... that his SAH might have been the result of some kind of black magic although he quickly decided he really thought that possibly ... I was just feeling anxious last night

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... denied depression or cognitive behavioral changes post-SAH only acknowledging physical deficits. Finally, although ... restless ... pt said he would like to take his time ... to regain optimal level of function ...". Signature, illegible.

Undated Follow-Up Note

" ... Hx & PE: ... weight 180. BP 120/80. Heart rate 78 ... 41 y/o man here for f/u of labs S/P SAH 05/10/03 ... discharged on 08/19/03 ... doing well since discharge ... c/o mild spasm in (L) shoulder at certain angles, pain in morning which disappears after morning... also c/o numbness 2nd to 4th fingertips ... pt to return to Clinic in 3 mos...". Signed, Dr. Goldfarb

07/03/03 Consultation Request

" ... Reason: decrease sodium ... hyponatremia due to polydypsia and HCTZ effect (increase prox tubule water absorption) ... continue free water restriction ...". Signature, illegible.

07/04/03 CT Scan of the Head

"... unenhanced CT scan of the orbit is performed in axial and coronal 3 mm sections ... mild (L) orbital proptosis is noted ... the frontal sinuses are hypoplastic ...".

07/06/04 Neurology Resident Follow-Up Note

"... 42 y/o male ... here for routine f/u ... pt has had no breakthrough seizures ... he admits to some memory problems related to stress ... has no problem w/ remembering names, addresses... Neuro examination remains unchanged ... illeg ... shall continue ... Phenobarbitone 30 mg q A.M., 20 mg k.o. b.i.d. ... f/u after 3 mos ...". Signature, illegible.

01/07/05 Neuro Note

" ... Hx & Findings: ... BP: 128/74 ...".

03/11/05 Neurology Resident's Follow-Up Note

"... Neurologic exam remains non-focal ... advised him strongly to quit smoking ... this notes states Phenobarbitol has been stopped ... the pt has had no seizures ...".

ANALYSIS

Relevant Definitions with Comment

As mentioned above, Mr. Singh had used Herbalife, a preparation containing ephedra species, a mixture of sympathomimetic amines on a daily basis, perhaps as long as a year prior to his subarachnoid hemorrhage. He also used it on the day of his subarachnoid hemorrhage.

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Sympathomimetic Amine and Intracranial Hemorrhage

A sympathomimetic amine is a compound that imitates some of the actions of the naturally occurring human sympathetic amines, norepinephrine, epinephrine and dopamine. Norepinephrine and epinephrine are referred to as the "adrenergic amines."

The adrenergic amines effect their actions by stimulating a receptor which then triggers a specific action. Adrenergic receptors are labeled α -1, α -2, β -1, β -2 and β -3. Stimulation of the α -1 receptor, present on blood vessels, including those in the brain produces constriction of those blood vessels and also promotes increased systemic blood pressure.

Ephedrine, phenylpropanolamine and others in the ephedra species group are sympathomimetic amines that have the capacity to, and do, directly and indirectly stimulate the α -1 receptor, producing vasoconstriction (narrowing of blood vessels)*.

Vasoconstriction is effected by muscular contraction in the vessel wall. Since the muscular sheeting in cerebral blood vessels is not continuous but, rather, is spiraled, α -1 stimulation produces irregular contraction patterns known as segmental constriction, although the segmental length can be variable.

It is obvious that segmental constriction will produce regional alteration in the velocity, volume, pressure and direction of blood flowing through the affected vessel and that jetting of flow will occur and that turbulence will be produced. These rheologic phenomena initially damage the inner lining of blood vessel walls (endothelium) and then the entire vessel wall. The regional rheologic alterations, vessel wall damage and systemic blood pressure effects described above are the prime mechanisms by which ephedra containing compounds such as Herbalife are associated with hemorrhagic strokes. These factors can act singly or in tandem to produce a hemorrhagic stroke. Thus, when there is sufficient cerebral vessel wall damage, the vessel will no longer be able to contain its contents (blood) and bleeding will ensue resulting in "intracranial hemorrhage".

The location of intracranial bleeding is determined by the site of the damaged vessel, and the direction of bleeding. Such intracranial bleeds are more specifically named by their location: intracerebral, intraventricular, subarachnoid, subdural and epidural. At times,

* Amphetamine, a synthetic compound derived from ephedra species, has been the most studied agent and serves as a model for this group.

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hemorrhage may be present in more than one location. All the factors above outlining the mechanisms and physical characteristics underlying ephedra containing products such as Herbalife's association with intracranial hemorrhage apply whether the vessels so exposed are of anomalous structure or not. The presence of the most typical anomalies, aneurysms (as in Mr. Singh's case) and arteriovenous malformations, simply render these structures more vulnerable to the deleterious effects of the pharmacologic mix contained in Herbalife.

Subarachnoid Space - Subarachnoid Hemorrhage

The subarachnoid space is a fluid (cerebrospinal fluid) filled sac encompassing the central nervous system and lies between the pial membrane, which intimately invests the brain and spinal cord, and the arachnoid membrane which overlies it. Since cerebral aneurysms tend to occur in vessels where they traverse the subarachnoid space, when they rupture, the extravasation of blood is into the subarachnoid space, hence "subarachnoid hemorrhage". Other than trauma, rupture of a cerebral aneurysm is the primary cause of subarachnoid hemorrhage.

Cerebral Aneurysm/Cerebral Aneurysmal Rupture

Cerebral aneurysms are out-pouchings in the walls of the arteries that supply the brain. There are many ways of classifying cerebral aneurysms, but in general they are considered to be congenital or of developmental origin, the classical saccular or "berry" aneurysm; or of arteriosclerotic origin the "fusiform aneurysm". Aneurysms may be of infectious origin, the "mycotic aneurysm" or the result of arterial dissection, trauma or more rarely of myxomatous or neoplastic origin. Cerebral aneurysms can be associated with fibromuscular dysplasia, a rare condition of unknown (possibly developmental) etiology which was putatively diagnosed in Mr. Singh's case. A developmental aneurysm is thought to arise from congenital or developmental defects in the walls of the cerebral arteries, principally the muscular and endothelial layers and the internal elastic lamina or possibly a defect in the vaso vasorum. At times, a combination of factors is operative in aneurysm formation, expansion and eventual rupture.

Once a saccular cerebral aneurysm has formed, the usual factors promoting its thinning, expansion and rupture are arteriosclerosis and blood flow effects. These can be considered "wearing down" factors, thus the peak age of saccular aneurysm rupture is 55 to 60 years; these aneurysms having had enough time for their walls to be thinned and expanded by wearing down processes. However, the natural history of most cerebral aneurysms is overwhelmingly to never rupture; out of the approximately 15 million

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cerebral aneurysms harbored in the U.S. population, there are only about 30,000 ruptures a year. The ultimate setting for rupture is aneurysmal wall strength insufficient to contain aneurysmal content. The critical step in this mismatch is a rheologic surge in systemic blood pressure or perianeurysmal blood flow, applicable most obviously in younger patients such as Mr. Singh where wear and tear are factors. In fact, in Mr. Singh's case, there is no evidence of atherosclerosis or sustained systemic hypertension.

The usual causes of acute systemic blood pressure elevation and/or regional flow surges are emotional stress, endocrine disturbances, renal vascular disease, surgical procedures about the head and neck, dental procedures, surgical procedures about the heart, hypertensive crisis in a chronic hypertensive, increased intracranial pressure, ingestion of toxins including alcohol and cigarette smoke, infection, neoplasm, pharmacologic agents such as ephedra species, eclampsia, aortic conditions and alteration in blood volume. Additional special circumstances can particularly influence the likelihood of aneurysmal hemorrhage. They include, turbulence of blood flow, jetting of blood flow, pregnancy, aneurysmal size, cigarette smoking on the day of rupture, binge alcohol drinking, disturbance of clotting mechanisms or failure of cerebral autoregulation.

Differential Diagnosis

Differential diagnosis is the standard method used in medicine to arrive at the cause (etiology) of, or factors associated with a defined clinical condition (syndrome). Simply stated, this logical process is performed by listing all the possible causes of the syndrome, then examining available clinical data insofar as they support or fail to support each differential possibility. If differential possibilities are not supported by data, they are considered "ruled out" or eliminated or allowing for the extremely unusual sometimes seen in medicine, "improbable". At times, more than one factor may be operative in the production of a clinical syndrome; there thus may be multiple contributing factors causing a particular syndrome.

CURRENT STATUS (11/14/05)

Mr. Singh suffers headaches of recent onset (within the last few months). These headaches occur every day and Mr. Singh rates the pain as "6 out of 10." The headaches may last up to two hours and are felt in the frontal region and the right side of his head. Mr. Singh uses Tylenol for pain relief. He states that occasionally, his headache pain feels "like [his] head is going to explode." Mr. Singh denies any warning or prodrome before the headaches ensue. There is associated photophobia and the headaches occasionally awaken him from sleep. Since sustaining his

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subarachnoid hemorrhage, Mr. Singh feels that his memory "doesn't work well" and he has difficulty expressing himself. Mr. and Mrs. Singh agree he is now emotionally labile and, by his own description "whines and cries", has become a hypochondriac and is impatient." Mr. Singh states he is depressed but has not had suicidal thoughts. In general, Mr. Singh has suffered a significant psycho-social change. Wherein he previously was a bold, strong engaging personality, he is now extremely fearful; he is "depressed, scared, everything frightens him" and he was become withdrawn. Additionally, since the subarachnoid hemorrhage, Mr. Singh lacks sexual desire and can't achieve erection. He states he had been sexually "very active" prior to his stroke. After recovering from his subarachnoid hemorrhage sufficiently to attempt ambulation, Mr. Singh had difficulty walking, which persists to this day. Currently, he feels that his legs are weak and that he is unsteady walking. Mr. Singh reports difficulty negotiating stairs, more so ascending than descending.

Mr. Singh has "hissing" tinnitus in both ears; he is unsure how long this has been present. Over the past year, Mr. Singh has developed neck and upper and lower back pain. Mr. Singh goes to a chiropractor in Manhattan, Dr. Martin, who administers manipulative treatment for these problems. Mr. Singh also notes that his hands are numb, particularly involving the 3rd, 4th and 5th fingers of both hands.

Mr. Singh lives with his wife in Queens in an apartment in an elevator building. However, he states that sometimes, during the week, they stay in an elevatorless rooming house in Manhattan. Mr. Singh does not do household chores but attends to his personal activities of daily living and grooming. Mr. Singh spends some of this time attempting to help his wife at work. He states he usually "walks all day long to kill time." Mr. Singh naps during the day; he watches television and reads but has difficulty concentrating. He uses a computer for recreation but not for extended periods of time. Mr. Singh no longer drives a car because he is afraid to drive. He previously worked full time but can no longer do so. Previously, Mr. Singh attended a gym two to three times a week, which he has curtailed because of his post-subarachnoid hemorrhage deficits. He no longer socializes or plays cards, activities which he previously enjoyed. Mr. Singh can use public transportation and travels independently.

Mr. Singh receives medical treatment at the St. Vincent's Clinic in Manhattan which he generally attends every other month. He is under the care of Dr. Sathi, a neurologist, whom he states prescribed multivitamins and vitamin E for "stiff joints." As noted above, Mr. Singh uses Tylenol for headaches, up to six times a day, but states he often doesn't need the Tylenol at all. Mr. Singh receives acupuncture once a month for back and neck pain in addition to chiropractic manipulation. Because acupuncture treatment is obtained at a school, he is treated by a number of different students.

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PAST MEDICAL HISTORY

Mr. Singh denies any significant previous illness. He sustained an injury to his right arm in childhood which required surgery. He denies the abuse of drugs and alcohol. He is allergic to penicillin. Until his 2003 subarachnoid hemorrhage, Mr. Singh had been a pack a day smoker for thirty years. He states he has subsequently stopped smoking. Mr. Singh was born in India and has lived in the U.S. for approximately fifteen years. Mr. Singh is a college graduate and is literate in English, Hindi and Punjabi. Mr. Singh worked in jewelry sales and repair; specifically he repaired silver jewelry. He denies any other personal or familial history.

NEUROLOGIC EXAMINATION (11/14/05)

GENERAL Mr. Singh is a well nourished, well developed right handed man who is 5'6" tall and weighs 184 pounds. Blood pressure is 131/82; pulse 74 and regular. Peripheral pulses are normal. Examination of the head and neck reveals no bruits, thrills or masses. There are no sweating deficits. There is no generalized wasting. The patient is appropriately disrobed for the examination. Mr. Singh has well healed surgical scars appropriate to his tracheostomy and feeding tube site. The right arm is attenuated although thicker in muscle bulk than the left; he holds the arm in forearm supination, secondary to the childhood injury noted above. There is a keloidal scar across the volar distal right forearm.

MENTAL STATUS Mr. Singh is awake, alert and oriented to person, place and time. Speech is fluent and there is no anomia. Thought content is non-delusional and relevant. General information is adequate. He makes many errors on mental status testing however. Mr. Singh is circumstantial, becomes catastrophic and even whimpers when pressed on examination. Mr. Singh cannot read or interpret more than simple sentences. He cannot read aloud. On the King story, Mr. Singh totally missed the point of the story nor could he retrieve details or keep the facts in order. Mr. Singh could not understand the following paragraph which he read both aloud and to himself: "Double Negatives: Avoid Double Negatives. Few of us would say not never, not no, not nothing, but remember that the adverbs hardly, rarely and scarcely are also considered negative and do not take a 'not'. So instead of say 'he can't hardly write', say, ' he can hardly write'." Even given time and prompting, Mr. Singh has difficulty with the rudiments of this sentence. He has difficulty crossing the midline. On simultagnosia testing, he cannot see the "4" made up of "3s;" he demonstrates constructional apraxia on attempting to draw a cube. The simultagnosia card is included in the chart with a sample of his handwriting.

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CRANIAL NERVES Cranial nerve functions II-XII are intact. The visual fields and fundi are normal. There are no wiring, narrowing or crossing defects, nor are there exudates, hemorrhages or microaneurysms. The discs are maculae are normal. Hearing and speech are intact.

GAIT Gait and associated movements are clumsy. He has a truncal tilt to the left and veers to the left when he walks. The right foot splays out and the left lower extremity is energetically circumducted. He does not tandem, heel or toe walk because he is fearful of losing balance. There is no Romberg sign.

POWER Power is adequate throughout and muscle appearance is normal. There is no focal atrophy or wasting. Tone is normal throughout and there are no fasciculations.

RAPID ALTERNATING/FINE MOVEMENTS On rapid alternating movements, toe tapping is poor bilaterally, right worse than left. Heel swiveling is also performed poorly, bilaterally. He clumsily beats out a tattoo with either hand, although gross upper extremity RAMs are adequate bilaterally.

CEREBELLAR There is no finger to nose or heel to knee dystaxia.

REFLEXES The deep tendon reflexes are obtainable and physiologic throughout. There is no clonus. Positive left Babinski sign. There are no primitive reflexes present.

RANGE OF MOTION There is no significant restriction of motion of the spine or extremities. There are no Spurling or Fabere signs.

MECHANICAL There is no visible or palpable paravertebral muscle spasm or percussion tenderness over the spine. No Tinel or Vallieux signs are elicited at the wrist, forearms or elbows.

SENSATION Sensation to pin and light touch is diminished over the left face and left arm and there are inconsistent errors to pin and touch throughout. Joint position and vibration are maintained.

MENINGEAL There is no nuchal rigidity, photophobia or eyeball tenderness. There are no Kernig or Brudzinski signs present.

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EXTRAPYRAMIDAL There is no rigidity, cogwheeling or adventitious movement noted.

The remainder of the neurologic examination is within normal limits.

DIAGNOSIS & CONCLUSION

1. Status post subarachnoid hemorrhage with intraparenchymal bleeding into the right lenticular nucleus and left frontal convexity, communicating hydrocephalus and ischemic injury to the right lenticular nucleus.
2. Status post electrocoagulation of left internal carotid aneurysm.
3. Probable fibromuscular dysplasia involving left internal carotid artery.

The resultant encephalopathy has produced severe permanent functional and anatomic brain damage with severe permanent impairment in cognitive, psychological and sexual functions with lesser but significant impact on Mr. Singh's ambulatory and motor function. He is permanently severely disabled in the fullest sense; he is unable to work at his usual profession and cannot enjoy the fruits of life.

APPLIED DIFFERENTIAL DIAGNOSIS

Consideration of the differential list of precipitants of aneurysmal rupture above in the light of review of Mr. Singh's medical records eliminates all precipitants except for his use of the ephedra containing product Herbalife. Cigarette smoking, in Mr. Singh's case, is a predisposing risk factor. Mr. Singh is not a hypertensive. The elevated blood pressure recorded on admission was the result, not cause of, his subarachnoid hemorrhage. After clinical stabilization, Mr. Singh's blood pressures were and remained in the ideal to normotensive range. Significantly, there are no degenerative changes described in the retinal vessels, which, are extensions of cerebral blood vessels that most clearly parallel cerebral vascular hypertensive or arteriosclerotic damage and there are no systemic indications of these conditions present. Thus, the usual wear and tear factors contributing to aneurysmal rupture are at best minimally present. That ephedra containing products such as Herbalife have been related to aneurysmal rupture and consequent subarachnoid hemorrhage is well demonstrated in the medical literature. (See bibliography.)

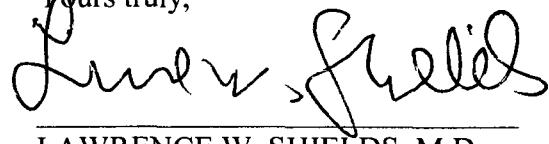
CONCLUSION

Thus, on the basis of the review of Mr. Singh's medical records, the employment of accepted pharmaco- physiologic principles and reference to conventional medical literature, it may be stated with a reasonable degree of medical certainty that Harbir Singh's use of Herbalife on the

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day of and the months preceding his subarachnoid hemorrhage on May 10, 2003, more likely than not substantially contributed to that subarachnoid hemorrhage and had Harbir Singh not used Herbalife as described above, more likely than not he would not have suffered subarachnoid hemorrhage on May 10, 2003, and sustained the neurologic deficits he now demonstrates. Cigarette smoking was a predisposing risk factor.

Yours truly,



MD

LAWRENCE W. SHIELDS, M.D.
Board Certified Neurologist

LWS:djk

HARBIR SINGH
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DATE OF BIRTH	March 4, 1940, New York City	
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RESIDENCY	Mount Sinai Hospital, New York, N.Y. Department of Neurology	7/68 - 6/71
GOVERNMENT SERVICE	U.S. Public Health Service Hospital, Lompoc, CA General Medical Officer	7/66 - 7/67
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LICENSURE	New York 100566 California G18595 Pennsylvania 042659E	1968 1970 1989

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American Medical Association
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American Academy of Neurology
American Academy of Disability
Evaluating Physicians - Fellow

APPOINTMENTS

Medical Consultant to the District
Attorney, Kings County

HOSPITAL AFFILIATIONS

Beth Israel Medical Center
New York, N.Y. 1987 - 2000

Long Beach Memorial Hospital
Long Beach, N.Y. 1981 - present

Peninsula Hospital Medical Center
Far Rockaway, N.Y. 1981 - present

New York Eye & Ear Infirmary
New York, N.Y. 1993 - 1995
Director - Neurology Unit

St. John's Episcopal Hospital
Far Rockaway, N.Y. 1981 - 1991

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MISCELLANEOUS	Albert Einstein College of Medicine Department of Psychiatry Visiting Psychiatrist	09/70 - 04/71
	Lincoln Hospital, Bronx, N.Y. Methadone Clinic Director	02/70 - 04/71
	New York City Medical Examiner's Office, New York, N.Y. Investigator	02/70 - 04/71
	New York City Department of Corrections, New York, N.Y. Clinician	07/68 - 04/71
	U.S. Public Health Service Hospital, Danbury, CT Federal Narcotics Addiction Rehabilitation Act Project Director	07/67 - 07/68

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	Bellevue Hospital Psychiatry Fellowship Management of the Unmanageable Patient: An inquiry concerning patients who apparently become psychotic of general medical and surgical wards.	02/63 - 08/63
	New York State Public Health Fellowship Suicide in Nassau County: An epidemiologic study.	05/62 - 09/62
CHIEF CURRENT INTERESTS	Clinical Brain Tumor Research Project Private practice of Neurology Medical/Legal Consultation	08/98 - 2000 1971 - present 1988 - present
MASS TORT LITIGATION EXPERIENCE	Have reviewed and rendered opinions in 500+ cases of use of ephedra-containing compounds and their potential neurologic complications.	
	Have been a consultant on welding rod related - manganese toxicity cases.	
	Lectured on Strokes and PPA, Ephedrine & Related Drugs, ATLA, Phoenix	06/2001
	Lectured on Strokes and PPA, Ephedrine & Related Drugs, ATLA, Dallas	10/2001

COURT APPEARANCES BY LAWRENCE W. SHIELDS, M.D.

<u>DATE</u>	<u>VENUE & ATTORNEY</u>	<u>CASE NAME</u>	<u>SUBJECT MATTER</u>
01/19/05	STATE SUPREME COURT COUNTY OF BRONX Sullivan, Papain, Block McGrath & Cannavo	Salvatore Palazzo	Medical Malpractice - Permanent disabling compli- cations following treatment for bile duct stones.
09/15/04	STATE SUPREME COURT Sullivan, Papain, Block, McGrath & Cannavo	Alice Huang	Personal injury case - injuries sustained when struck and dragged by train
04/14/04	STATE SUPREME COURT COUNTY OF QUEENS Sullivan, Papain, Block, McGrath & Cannavo	Theodore Monte	Personal injury - Injured when responding to fire caused by improper repair causing collapse of ceiling.
03/18/04	STATE SUPREME COURT COUNTY OF KINGS Edward Suh & Assoc.	Chun McKinnie	Personal injury case - woman struck and dragged by minibus in crosswalk.
02/09/04	STATE SUPREME COURT COUNTY OF SUFFOLK Steven M. Goldfarb trial counsel to Ronald Roth	Lillian Bedell	Medical Malpractice - injury to peroneal nerve.
01/21/04	STATE SUPREME COURT Sullivan, Papain, Block, McGrath and Cannavo	Joseph Cuiillo	Injuries sustained in MVA with complications and eventual demise.

COURT APPEARANCES BY LAWRENCE W. SHIELDS, M.D.

<u>DATE</u>	<u>VENUE & ATTORNEY</u>	<u>CASE NAME</u>	<u>SUBJECT MATTER</u>
09/ /03	STATE SUPREME COURT Indiana Dennis Hartley, Esq.	Jason Davidson	Murder defense involving limbic system frontal lobe injury impact of drugs in in brain and behavior.
07/16/03	STATE SUPREME COURT COUNTY OF RICHMOND (N.Y.) Sullivan, Papain, Block, McGrath & Cannavo	Gloria Narvaez	Malpractice case involving carotid cavernous fistula.
07/21/03	STATE SUPREME COURT COUNTY OF BRONX (N.Y.) Wilson, Elser, Moskowitz Edelman & Dicker	Peter Chisolm	Defense case involving trauma to ulnar nerve neuropathy.
07/01/03	STATE SUPREME COURT COUNTY OF QUEENS (N.Y.) Shaw, Licitra, Bohner, Esernio, Schwartz & Pfluger	Eun-Hye Kang	Personal injury case involving traumatic brain injury.
06/24/03	STATE SUPREME COURT (N.Y.) Kennedy & Associates Thomas G. Nolan, Of Counsel	Marc Nascimento	Personal injury case - wrongful death case concerning conscious pain and suffering.

COURT APPEARANCES BY LAWRENCE W. SHIELDS, M.D.

<u>DATE</u>	<u>VENUE & ATTORNEY</u>	<u>CASE NAME</u>	<u>SUBJECT MATTER</u>
06/16/03	STATE SUPREME COURT COUNTY OF KINGS (N.Y.) Kramer, Dillof, Livingston and Moore	Thomas Spero	Malpractice - baby birth injury case with Erb's Palsy and perinatal brain damage.
06/17/03	STATE SUPREME COURT (N.J.) Nagel, Rice, Dreifuss and Mazie	Sayonara Bhattachayra	Deposition - Ephedra caused stroke.
06/04/03	STATE SUPREME COURT (Tx) Waters & Krause	Raul Cardenas	Deposition - ruptured cerebral aneurysm - PPA related.
05/02/03	STATE SUPREME COURT (N.Y.) Shaw, Licitra, Bohner et al.	David Fellin	Malpractice - Ruptured cerebral aneurysm, missed diagnosis.
04/22/03	STATE SUPREME COURT (MS) Brian Herrington	Todd McKinley	Deposition - PPA related stroke.
03/27/03	FEDERAL COURT Southern District, NY Sullivan, Papain, Block, McGrath & Cannavo, P.C.	James Peebles	Testimony - Personal Injury.

COURT APPEARANCES BY LAWRENCE W. SHIELDS, M.D.

<u>DATE</u>	<u>VENUE & ATTORNEY</u>	<u>CASE NAME</u>	<u>SUBJECT MATTER</u>
11/15/02	STATE SUPREME COURT (N.Y.) Shaw, Licitra, Bohner et al.	Ghazi Bokhari	Malpractice - diagnosis and treatment of tuberculous epidural abscess.
11/14/01	STATE SUPREME COURT* Salenger & Sack	Altaf Ali	Medical malpractice; sciatic nerve palsy secondary to operative mishap.
03/12/01	STATE SUPREME COURT* Shandell, Blitz, Blitz Bookson & Kern	Sandra Welch-Todd	Medical malpractice; wrongful death secondary to seizures.
12/12/00	STATE SUPREME COURT* Kramer, Dillof, Tessel Duffy & Moore	Alexandria Ramos	Perinatal hypoxic ischemic injury.
10/31/00	STATE SUPREME COURT* Sullivan, Papain, Block McGrath & Cannavo David Dean	Nandranie Rameswar	Motor vehicle accident; pedestrian struck. Post-traumatic cervical and lumbar radiculopathy.
09/26/99	STATE SUPREME COURT* Levy, Phillips & Konigsberg	Eva Leopold	Medical malpractice; Post-operative brain stem injury.

COURT APPEARANCES BY LAWRENCE W. SHIELDS, M.D.

<u>DATE</u>	<u>VENUE & ATTORNEY</u>	<u>CASE NAME</u>	<u>SUBJECT MATTER</u>
06/18/99	STATE SUPREME COURT * Kramer, Dillof, Tessel Duffy & Moore	Roberto Leyton	Traumatic brain injury; pain, suffering (records only)

* Denotes Supreme Court State of New York but not specific county.